MEDICAL INTELLIGENCE

CONCENTRATIONS OF NICOTINE AND TOBACCO SMOKE IN PUBLIC PLACES

WILLIAM C. HINDS, SC.D.,
AND MELVIN W. FIRST, SC.D.

PUBLIC interest has focused on health effects to the large numbers of nonsmokers exposed to tobacco smoke in public places. Recent regulations in a few cities have banned smoking in public places, or have restricted smoking in the manner of United States commercial aircraft.

Two studies[1,2] indicated that in crowded private rooms concentrations of tobacco smoke often exceed 260 μg per cubic meter, the federal air-quality standard for particulate matter that is not to be exceeded more than one day per year. Hoegg[1] estimated that in residences, meeting rooms, or private automobiles, the nonsmoker inhales in one hour the equivalent of smoking 0.01 to 0.20 cigarettes. Bridge and Corn,[2] by measuring carbon monoxide during party situations involving 50 to 73 people in rooms of 140 and 100 m^3 under controlled ventilation conditions, estimated smoke concentrations to be 2000 to 4000 μg per cubic meter and concluded that these levels are a matter of concern.

Estimation of levels of tobacco smoke in public places was undertaken to evaluate the health implications for nonsmokers. Measurements were limited to the particulate phase of tobacco smoke, although it is known that the gaseous phase also contains substances that may affect health. Since the objective was to measure only tobacco smoke, all methods commonly used to measure total suspended particulate matter were ruled out because of the many other sources of particulate matter in the indoor atmosphere. The use of carbon monoxide as a tracer has similar disadvantages because of the widespread distribution of this common air pollutant. Nicotine was chosen as the tracer for tobacco smoke for the following reasons: it is specific for tobacco smoke (the only other source of nicotine is from agricultural sprays, which are unlikely to be a contaminant of the indoor atmospheres tested); with the exception of water, nicotine is the largest single component of the particulate phase of tobacco smoke; nicotine concentration is unaffected by the moisture content of the smoke; and sensitive gas chromatographic analytical methods are available for measurement of nicotine concentrations.

From the Department of Environmental Health Sciences, Harvard School of Public Health (reprint requests should be addressed to Dr. Hinds at the Harvard School of Public Health, 665 Huntington Ave., Boston, MA 02115).

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Because of the wide range of public places evaluated and the small number of samples, the procedures employed and the results should be considered a pilot study having the limited objective of defining the extent of the "passive-smoking" problem in public places.

SAMPLING METHODS

The procedure was to enter a public place as a patron and sample a known volume of air through an AA Millipore filter having a collection efficiency for tobacco smoke greater than 99 per cent. Samples were taken with an inconspicuous battery-powered pump at a rate of 4 liters per minute for a maximum period of 2½ hours. The entire sampling system weighed 1.3 kg and was contained in a phenolic box, 17 by 13 by 6 cm (Fig. 1). To obtain realistic samples, the unit was placed as close to the breathing zone as possible — e.g., on a table in a restaurant, or on a lap in a train.

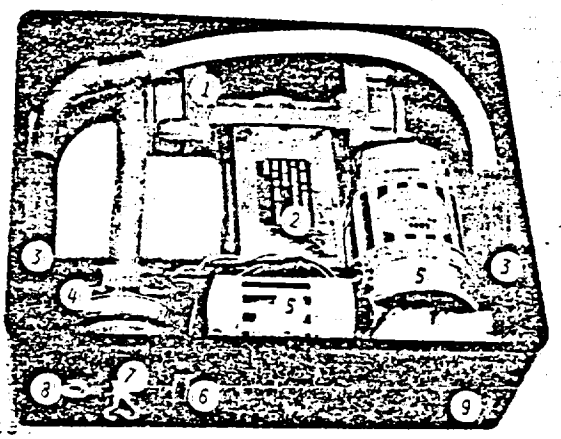

Figure 1. The Sampling System, Showing the Pump (1), Motor (2), Pulsation Damper (3), Filter Holder (4), Rechargeable Batteries (5), On-Off Switch (6), Air Inlet (7), Battery-Charging Jack (8), and Case (9).

The material trapped on the filter was extracted with distilled water, concentrated by rotary evaporation, and analyzed for nicotine with a gas chromatographic technic described by Jacin et al.[3] The nicotine content was used to calculate the tobacco-smoke particulate concentration on the basis of an experimentally determined nicotine fraction of 2.6 per cent established by measurement of total particulate mass and nicotine concentration of sidestream smoke in an aerosol chamber. Sidestream smoke is the principal component of indoor tobacco-smoke pollution (i.e., 80 to 90 per cent).[1,2]

Tests were run with filter and nonfilter cigarettes, and current sales figures[4] were used to calculate the weighted average nicotine fraction as 2.6 per cent. No noteworthy concentration effect on nicotine fraction was observed for smoke concentrations ranging from 6000 to 110,000 μg per cubic meter. Our ambient measurements were an order of magnitude smaller than this range.

Twenty-three samples were taken in the Boston area during 1973 and early 1974. Some types of public areas — commuter trains, commuter buses, and bus and airline waiting rooms — were sampled repeatedly, whereas others, such as large, crowded restaurants and lounges, are represented by individual samples. On buses and trains no attempt was made to sample in smoking or nonsmoking sections because these designations are largely ignored by passengers.

RESULTS

Smoke concentration for each category of public place is shown in Table 1 as weight per unit volume of sampled

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SMOKING AND NONSMOKERS — WHAT IS THE ISSUE?

With tobacco consumption near an all-time high, a fervent interest has developed over the effect of passive smoke inhalation on the nonsmoker. Controversy exists on the rights of the nonsmoker to "clean" air versus the rights of the smoker to enjoy a product consumed by man for centuries. This issue extends far beyond the health professions and is complicated by the divergent interests of many groups, with approximately 160 proposals to restrict or segregate the smoker now pending before legislatures in 39 states. The primary issue is how much tobacco the nonsmoker passively inhales and what the potential health hazards of such exposure are. The number of publications attempting to answer these questions is very large. Until the contribution by Hinds and First in this issue of the *Journal*, reliable data on relevant public passive exposures to tobacco smoke have been sparse.

When cigarettes are consumed, tobacco smoke is generated in part as mainstream smoke, essentially all of which is actively inhaled and 70 to 90 per cent of which is retained by the smoker. Sidestream smoke of the burning cigarette is the primary source of exposure to the nonsmoker, representing about 50 per cent of the tobacco burned and responsible for approximately two thirds of the aerosol particles delivered to the environment. Smoke contains about 2000 or more identifiable components and can be divided into gas and particulate phases.[1] Ambient concentrations of trace components can be measured, and potential amounts of pyrolized products available for inhalation by the nonsmoker determined.

Using nicotine as a tracer of the particulate phase, Hinds and First have indicated that the nonsmoker can potentially inhale only extremely small amounts of tobacco smoke. Nicotine may be an ideal tracer for such studies, since it is basically unique to tobacco. Other investigations have indicated that nicotine may occur in the urine of nonsmokers in concentrations at approximately 5 per cent that of smokers.[1] Contrary to the conclusions of Hinds and First, this figure suggests a passive consumption by the nonsmoker of about one full cigarette over an hour. These results, however, were obtained under experimentally high concentrations intolerable to prolonged human exposure. Carbon monoxide, a gas-phase component, has been used as a popular alternative tobacco tracer, producing environmental concentrations of 10 ppm or so.[2] For comparison, concentrations of 30 ppm are not uncommon in dense urban automobile traffic, and 100 ppm can be reached in polluted cities with temperature inversions.[2] Regardless of how studies are performed, only limited conclusions can be achieved by employment of tracers.

Definitive answers to questions of tobacco and nonsmokers should be obtained from data demonstrating the presence or absence of potentially associated diseases in man. That kind of information is extremely hard to come by. Potential health effects of tobacco on the nonsmoker have recently been reviewed[3,4] and are summarized as follows. Acute effects of short-term exposure to environmental tobacco smoke in population studies have not been evaluated in adequate depth. No chronic studies have been reported for adults. No data are available to demonstrate health effects of physiologic responses to nicotine levels reached in adult nonsmokers, and carbon monoxide concentrations in nonsmokers are far below levels that are of known health hazard. Potential effects of other smoke components on nonsmokers are conjectural. Information is lacking on cumulative effects of prolonged passive exposure to tobacco-smoke products. Potential additive or synergistic effects of tobacco-smoke products with nontobacco environmental contaminants need to be investigated. Interesting data have appeared, however, to suggest an increasing prevalence of acute respiratory disease in young children whose parents smoke.[5,6] These studies have been criticized because the data are sparse and perhaps influenced by socioeconomic and housing conditions, infections in parents, genetic differences and other factors.[7]

Hypersensitivity reactions to tobacco in the nonsmoker deserve more clarification. Commonly, atopic persons complain that tobacco smoke provokes respiratory-tract symptoms. Many agents in tobacco smoke could theoretically act as haptens and produce immunologic responses after binding to suitable carriers, but there is little experimental or epidemiologic evidence that sensitization to tobacco smoke exists.[8] Fletcher et al., in a report to the Royal College of Physicians of London, concluded that "...there is no evidence that other people's smoke is dangerous to healthy nonsmokers, but it can be extremely irritating and cause distressing symptoms, especially in allergic persons or in those already affected by heart or lung disease."[9]

One is left, then, with a perplexing and unsolved dilemma. The data of Hinds and First demonstrate that in public places nonsmokers could potentially consume 1/1000 to 1/100 of one filter cigarette per hour, a level of exposure that has had no known serious association with disease. Why, then, do so many nonsmokers appear to be adversely affected by passive exposure to tobacco? Perhaps acrolein and aldehydes, present in the smoke, act as irritants in extremely low concentrations and especially affect latently or overtly hypersensitive persons. The odor threshold for stale smoke components, which is also very low, may trigger emotional responses not yet well understood.

Clearly, numerous nonsmokers feel impaired in their well-being if exposed to tobacco smoke. The World Health Organization defines health as a condition characterized not only by the absence of diseases and infirmity but also by the presence of full psychologic, mental and social well-being.[10] By this definition, the nonsmoker passively exposed to tobacco may indeed have an adverse health response on a psychogenic basis. The issues remain unanswered beyond that, and, to solve this problem, scientific contributions in this area should be broadened, thereby diminishing speculation. In developing crucially needed research on this issue, one should identify the compounds of tobacco smoke that provoke adverse reactions in nonsmokers, establish if these reactions are dose

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